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Original Communication

Postmortem diagnosis of acute haemorrhagic pancreatitis

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ABSTRACT

Forensic pathologists can help in the investigation of sudden unexpected deaths in co-operation with the officials responsible for the maintenance of law and order to administer justice. Sudden unexpected deaths form the subject of medicolegal investigation if they occur in apparently healthy individuals, wherein an autopsy would shed light regarding the cause of death. A 4 year retrospective review of autopsy files at the Department of Forensic Medicine, Kasturba Medical College, Mangalore, South India was undertaken for cases of sudden unexpected deaths due to acute haemorrhagic pancreatitis occurring between May 2004 and April 2008. A total of seven cases of acute haemorrhagic pancreatitis diagnosed at autopsy as the cause of sudden unexpected death during the study period are discussed herein.

1. Introduction

One of the most difficult tasks for a forensic pathologist is opining the cause of death in sudden, unexpected, unwitnessed and medically unattended deaths.[1] In such circumstances, a forensic pathologist would not be able to opine with certainty that the death was natural in manner unless a full autopsy is performed. Sudden deaths are mainly attributed to the cardiovascular system. 45% of sudden deaths are related to the cardiovascular system, 25% to the respiratory system, 20% to the nervous system and 10% due to other causes.[2] In such deaths, a detailed autopsy may ultimately prove or disprove any allegations, which may be of significant value to the investigating authority. Sudden deaths due to acute haemorrhagic pancreatitis are comparatively uncommon. Herein, we describe a medicolegal autopsy case series of sudden unexpected deaths due to acute haemorrhagic pancreatitis.

2. Materials and methods

The autopsy files at the Department of Forensic Medicine, Kasturba Medical College, Mangalore, South India were reviewed for sudden unexpected deaths over a 4 year period from May 2004 to April 2008. In addition, the related police records were evaluated. The cases of sudden unexpected death due to acute haemorrhagic pancreatitis diagnosed at autopsy were identified. The age and sex of the deceased, circumstances of the death and autopsy findings of

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these cases forming the cohort of the study were summarized. The Department of Forensic Medicine, Kasturba Medical College, Mangalore, South India caters to around 90% of the autopsy services in Mangalore City at the Government Wenlock District Hospital, Mangalore.[3]

A total number of 2515 cases were autopsied during the period of 4-year period, of which 274 cases (10.89%) were death of sudden unexpected origin. In these cases, cardiac causes were the most common entity (102 cases; 37.27%), followed by respiratory causes (73 cases; 26.64%). Gastro-intestinal related causes (29 cases; 10.58%) and the central nervous origin causes (24 cases; 8.76%) were the least. 46 cases (16.79%) were grouped as miscellaneous which included malignancies, septicemia, etc. Of the sudden deaths due to gastro-intestinal origin (29 cases), 7 cases were due to acute hemorrhagic pancreatitis (24.14%) which are discussed herein. Overall, 2.5% of sudden unexpected death cases autopsied during the study period showed acute haemorrhagic pancreatitis. The cause of death was opined as acute haemorrhagic pancreatitis in only 0.3% of the total number of cases autopsied during the study period.

3. Results

We studied seven cases of acute haemorrhagic pancreatitis during the 4-year period. Six of them were males and all were in the age ranging from 21 to 58 years. Four of the six males were known alcoholics. The only female was aged 56 years. The overall mean age was 35 years. One male and one female were known diabetics. All cases presented as acute onset of symptoms leading to death. All the cases described in our study had an oedematous pancreas with haemorrhagic infiltrate (Fig. 1) and local inflammation of the peritoneal sac and large haematoma or abdominal haemorrhage in the retro-peritoneal space (Fig. 2). In all the cases, histopathology was suggestive of acute haemorrhagic pancreatitis. Toxicological analyses were negative in all the cases except one.

3.1. Case details

3.1.1. Case one

A 36-year-old male after a binge of alcohol was brought dead to the emergency department of the hospital. External findings yielded no clue as to the cause of death. On postmortem examination, peritoneal sac was inflamed and haemorrhagic in the pancreatic area with abdominal haemorrhage in the retro-peritoneal space. The pancreas weighed 110 g and was oedematous, with areas of haemorrhage. There was no evidence of gallstones, infections or obstruction of the hepato-biliary and pancreatic ducts. The other internal organs were congested. Pulmonary oedema and cerebral oedema were present. Histopatholgical sections of the pancreas showed extensive necrosis, haemorrhage and complete infarction of the parenchyma (Fig. 3). Lungs showed features of shock lung.

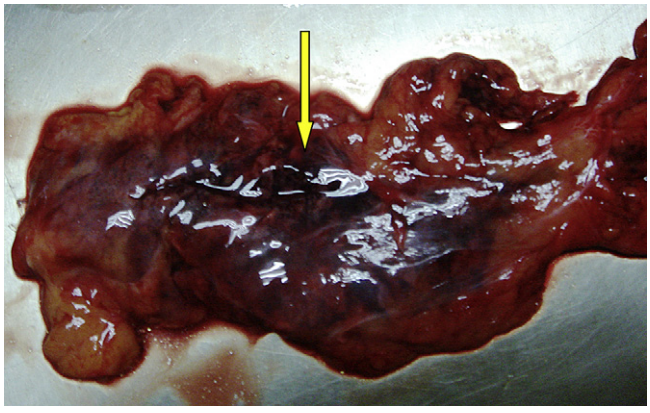

Fig. 1. Oedematous pancreas with haemorrhagic infiltrate.

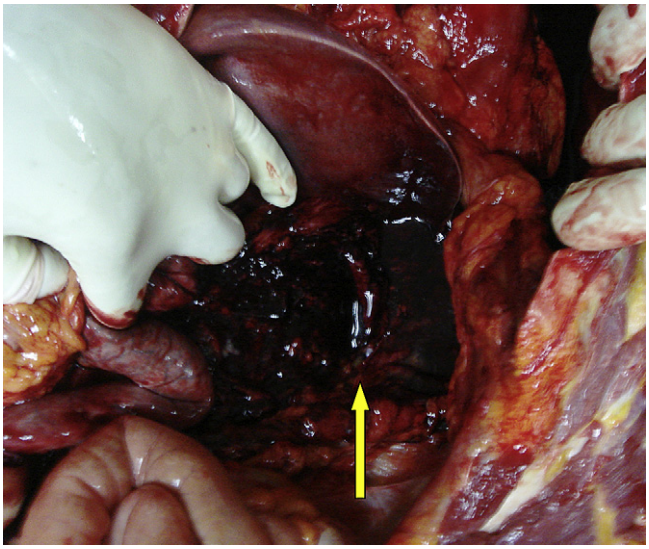

Fig. 2. Haematoma or abdominal haemorrhage in the retro-peritoneal space.

3.1.2. Case two

A 58-year-old male, a known diabetic patient, complained of chest pain and died on the way to the hospital. He used to consume alcohol regularly. At autopsy, the pancreas was oedematous and friable weighing 150 g with peripancreatic tissue haemorrhage and large haematoma in the retro-peritoneal space. Mild atheromatous changes were present in both the coronaries. However, there was no evidence of myocardial ischaemia. The brain and lungs were oedematous. Histopathology of the pancreas was consistent with acute haemorrhagic pancreatitis.

3.1.3. Case three

A 50-year-old male, alcoholic with recent past history of malaria was found dead in his room. There were no external injuries. On postmortem examination, all the organs were congested. Stomach contained partially digested food with alcoholic odour. The

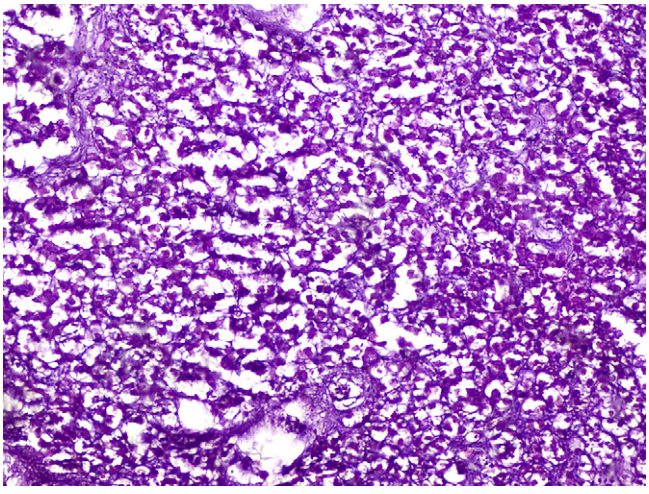

Fig. 3. Photomicrograph showing extensive necrosis and haemorrhage of the pancreatic acini and interstitium (Haematoxylin and Eosin × 200).

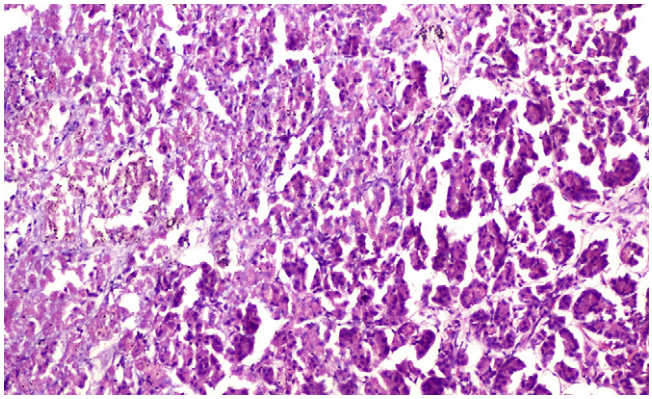

Fig. 4. Patchy necrosis of the pancreatic parenchyma with normal pancreatic acini on the right half (Haematoxylin and Eosin × 200).

pancreas weighed 120 g and was oedematous and haemorrhagic with abdominal haemorrhage in the retro-peritoneal space. Pulmonary oedema and cerebral oedema were present. Toxicological analysis of postmortem blood was positive for an unremarkable amount of ethyl alcohol. Histopathology sections from the pancreas showed patchy areas of haemorrhage, inflammatory infiltrate and infarction with intervening normal pancreatic acini (Fig. 4). Focal areas of ductular dilatation with necrosis of the ducts and surrounding interstitium were seen (Fig. 5). Lungs showed features of shock lung.

3.1.4. Case four

A 25-year-old male with history of chest pain was found dead in the morning. He was not a diabetic nor had any addictions. At autopsy, external examination was unremarkable. On internal examination, the stomach was empty. The pancreas weighed 110 g and was soft, friable and haemorrhagic. Abdominal haemorrhage was present in the retro-peritoneal space. All the organs were congested. Diffuse subarachnoid haemorrhage was present. The brain and lungs were oedematous. Histopathology sections of the pancreas showed complete infarction of the pancreatic lobules with haemorrhagic necrosis. The cause of death was certified as intracranial haemorrhage due to rupture of arterio-venous malformation with acute hemorrhagic pancreatitis, a natural mannered death.

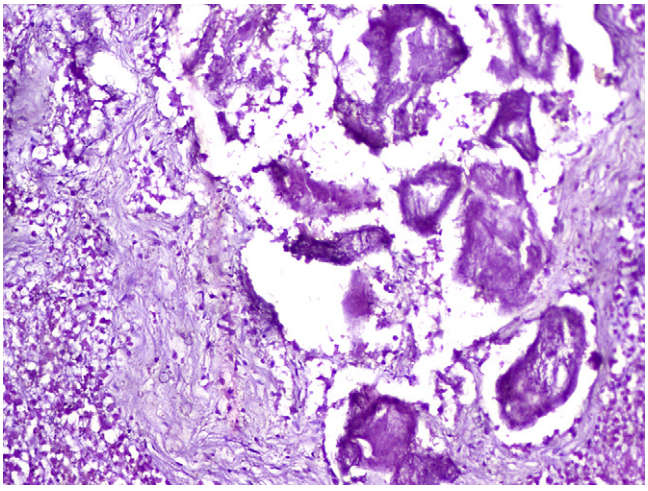

Fig. 5. Focal ductular dilatation with necrosis of the ducts and surrounding interstitium (Haematoxylin and Eosin × 200).

3.1.5. Case five

A 27-year-old male was found dead while on night duty in a hospital.[4] He did not have any addictions. At autopsy, external examination was unremarkable. Internal examination revealed an empty stomach. The gastric mucosa was congested with patchy erosions. The pancreas weighed 110 g and was soft, friable and haemorrhagic with a large haematoma in the retro-peritoneal space. Spleen, liver and the kidneys were congested. The brain and lungs were oedematous. Histopathology of the pancreas revealed haemorrhagic necrosis. The cause of death was certified as acute haemorrhagic pancreatitis, a natural mannered death. This case was previously reported.[4]

3.1.6. Case six

A 56-year-old female with sudden onset of severe abdominal discomfort was brought dead to the hospital. She was a known diabetic on regular treatment. At autopsy, external examination was unremarkable. On internal examination, the stomach was empty. The pancreas weighed 100 g and was soft, friable and haemorrhagic with abdominal haemorrhage in the retro-peritoneal space. All the organs were congested. The brain and lungs were oedematous. Toxicological analysis was negative. Histopathology of the pancreas revealed haemorrhagic necrosis. The cause of death was certified as acute hemorrhagic pancreatitis, a natural mannered death.

3.1.7. Case seven

A 37-year-old male, an alcoholic was found dead in his room. At autopsy, external examination did not reveal any injuries. On internal examination, the stomach was empty. The pancreas weighed 100 g and was spongy, friable and haemorrhagic with abdominal haemorrhage in the retro-peritoneal space. All the organs were congested. The brain and lungs were oedematous. Toxicological analysis was negative. Histopathology of the pancreas revealed haemorrhagic necrosis of the pancreatic lobules. The cause of death was certified as acute haemorrhagic pancreatitis, a natural mannered death.

4. Discussion

Acute haemmorghic pancreatitis is relatively a common disorder with an incidence rate of 10–20 cases per 1 lakh in western countries.[5] Approximately about 4.9% of patients who visited hospitals with a complaint of acute abdominal pain were found to have acute pancreatitis.[6] Thus, acute pancreatitis should be considered as a differential diagnosis for cases with signs and symptoms of digestive diseases. Acute pancreatitis is diagnosed based on the diagnostic criteria for acute pancreatitis proposed by the Research Committee of Intractable Diseases of the Pancreas.[7] Clinical criteria for the diagnosis of acute pancreatitis are acute abdominal pain and tenderness in the upper abdomen, elevated pancreatic enzyme levels in blood, urine, or ascitic fluid, and radiologic abnormalities characteristic of acute pancreatitis. Acute pancreatitis can be diagnosed when two or more of the above criteria are fulfilled and other causes of acute abdominal pain are excluded. Acute exacerbation of chronic pancreatitis is also included under the entity of acute pancreatitis.

A large retrospective autopsy study[8] of patients was analyzed to evaluate the major etiologic and pathologic factors contributing to fatal acute pancreatitis. From an autopsy population of 50,227 patients, 405(0.81%) cases were identified where acute pancreatitis was the primary cause of death. Acute pancreatitis was classified according to morphological and histological criteria, but not biochemical criteria. Patients with acute pancreatitis died significantly earlier than a control autopsy population of 38,259 patients.

The male:female ratio was 1.7:1 and the mean age was 52 years (range, 30–91 years). In our study, the age of the study sample ranged from 21 to 58 years with a mean of 35 years. The male: female ratio was 6:1 in our case series.

The clinical syndrome of acute pancreatitis and its complications have been well recognized. Although much has been known regarding the risk factors, pathology and biochemical events, the exact trigger events or pathogenesis still remains obscure. Major etiologic groups responsible for acute pancreatitis are chronic alcoholism and common bile duct stones with a small miscellaneous group including postabdominal surgery, viral hepatitis, drugs, postpartum cases, and the idiopathic group. 80% of cases are associated with alcoholism and billiary tract disease, and 10–20% are idiopathic.[5] Other causes include drugs (azathioprine, sulfonamides, sulindac, tetracycline, valproic acid, didanosine, methyldopa, oestrogens, furosemide, 6-mercaptopurine, pentamidine, 5-aminosalicylic acid compounds, corticosteroids, and octreotide), postERCP, infections (mumps, Epstein-Barr, coxsackievirus, echovirus, varicella-zoster, measles, mycoplasma pneumoniae, salmonella, campylobacter, mycobacterium tuberculosis, HIV), hypercalcaemia, hypertriglyceridemia, toxins and autoimmune pancreatitis.[9] The cases mentioned in the present study showed alcoholism as the most common etiological factor and two cases were suggestive of idiopathic variant.

The prevalence of established diabetes mellitus in the acute pancreatitis group was significantly higher than that observed in the autopsy control series.[8] In the present study, there were two cases of diabetics who were on treatment, suggesting that this disease should be considered as an additional etiological factor influencing survival in acute pancreatitis.

The infinite spectrum of pancreatitis severity is usually subdivided into mild and severe categories. In mild cases, the pancreas exhibits interstitial oedema and an inflammatory infiltrate without haemorrhage or necrosis, usually with minimal or no organ dysfunction. In severe cases, extensive inflammation and necrosis of the pancreatic parenchyma are present, often associated with severe gland dysfunction and multiorgan system failure.[10]

A myriad of systemic and local complications of acute necrotizing pancreatitis may occur. Local complications include gastrointestinal bleeding, infected necrosis leading to multiple organ failure and adjacent bowel necrosis.[11] Systemic complications include acute respiratory distress syndrome, acute renal failure, shock, coagulopathy, hyperglycaemia and hypocalcaemia.[12] Local involvement of the transverse colon can be a devastating complication.[13] Splenic vein thrombosis leading to variceal bleed, usually an under reported complication of acute pancreatitis accounts for the mortality in about 15% of cases.[14] The frequency of vascular necrosis in the form of pseudoaneurysm formation is in the range of 10%.[15] Life threatening heammorhage into the gastro-intestinal tract, retroperitonem, peritoneal cavity occurs in only 1–3% patients of acute pancreatitis but has a mortality of 50–80%.[16] Our study showed haemorrhage in the peritoneal cavity in all the cases.

Acute pancreatitis can be a mild, transitory illness or a severe, rapidly fatal disease. Acute inflammation of the pancreas is accompanied by the formation of necrotic areas on the surface of the pancreas and in the omentum and, frequently, also accompanied by haemorrhages into the substance of the gland. About 80% of cases of the disease are acute interstitial oedematous pancreatitis which have a low morbidity and mortality rate (<1%) and roughly 20% of patients with acute pancreatitis develop necrosis of pancreatic and peripancreatic tissues.[10] All the cases described in our study had an oedematous pancreas with haemorrhagic infiltrate and local inflammation of the peritoneal sac.

The study of 44 cases of severe bleeding following acute pancreatitis during a 10 year period reported the overall mortality rate to be 34.1%.[17] Splenic artery, portal vein, spleen, and unspecified peripancreatic vessels were the most commonly involved sources of bleeding, with associated mortality rates of 33.3%, 50%, 30% and 28.5% respectively.[17] Massive haemorrhage was more frequently associated with severe necrosis, with a mortality rate of 37.9%.[17] The present study too showed features of massive haemmorhage and necrosis in all the cases, implicating it as one of the major associations with sudden death.

Currently, infected pancreatic necrosis is still the leading cause of death related to diseases of the pancreas. Despite advances in monitoring systems and intensive care units early and worsening multiple organ failure still account for almost 40–50% mortality.[18,19] The morbidity and mortality associated with acute pancreatitis are substantially higher when necrosis is present, especially when the area of necrosis is also infected.[20] Almost 20% of patients with acute pancreatitis develop pancreatic necrosis.[21] A death rate of 1.8% in patients with sterile acute necrotizing pancreatitis and 24% in patients in patients with infected necrosis was observed.[22] The prevalence of pulmonary complications in autopsy studies ranged from 20% to 100%.[8,23,24] The most common extra pancreatic finding described is pulmonary oedema (49–81%). Studies have reported autopsy findings including pleural effusion (25–35%), bronchopneumonia (20–25%), atelectasis (25%), and pulmonary emboli in cases of fatal acute pancreatitis.[8,25,26] In our study, all seven cases had pulmonary oedema with histological features of shock lung in two cases. One of the cases had left sided haemorraghic pleural effusion. Cerebral oedema with subarachnoid haemorrhage was described at autopsy in cases of acute fatal pancreatitis.[8] In our study, diffuse subarachnoid haemorrhage was present in one case and cerebral oedema was present in all the cases. As per a study by Nashelsky and Lawrence[27] most presumed and actual causes of death were cardiovascular (94% and 80%, respectively) lacked features of a more specific cardiovascular process. The actual causes of death demonstrated a large breadth of cardiovascular and non-cardiovascular disease processes, even though ischaemic heart disease accounted for 62% of deaths. The presumed cause of death was completely wrong in 28% of the cases. This study demonstrates that doctors may generate erroneous death certificates for cases that are not autopsied. According to three studies, 30.2–50% of deaths due to acute pancreatitis were diagnosed at autopsy.[28–30]

There are relatively few reports of sudden death without symptoms due to acute haemorrhagic pancreatitis. Toffler and Spiro reported nine cases wherein patients were admitted to the hospital with shock or coma, and acute haemorrhagic necrotizing pancreatitis was recognized only at autopsy.[31] Di Maio and Di Maio described autopsies of individuals dying of natural disease, and found that 0.2% of their cases showed acute pancreatitis.[32] In the present study, 2.5% of sudden unexpected death cases autopsied showed acute haemorrhagic pancreatitis.

At autopsy, the most important critical diagnostic difficulty is postmortem pancreatic autolysis. The rapid onset of postmortem autolysis interrupts the histological recognition of the typical changes of pancreatitis, rendering extensive areas of devitalised tissue. The autopsy specimen often reveals 'mush' rather than typical acinar architecture. Inflammatory infiltrate, fat necrosis and calcium deposit are the most important pathologic features found in acute haemorrhagic pancreatitis by light microscopy, distinguishing acute haemorrhagic pancreatitis from postmortem pancreatic autolysis.[33] To avoid the diagnostic dilemma, the best time to acquire postmortem specimens from the pancreas for histological examination is up to eight hours after death. However, acceptable samples may be taken up to 12 h.[34]

Data derived from medicolegal autopsy studies should be included in future population-based studies of acute pancreatitis which would enable better understanding of the disease pathology

and possibly ensure timely detection and management of this potentially increasing cause of sudden death.

Conflict of interest

None declared.

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Ethical approval

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